01jul01 12:23:23 User208600 Session D1403.1

File 155:MEDLINE(R) 1966-2001/Jul W2 (c) format only 2001 Dialog Corporation

Set Items Description

Ref Items RT Index-term THYROIA E1 **E2 THYROICYTES E3** 90754 *THYROID 0 1 THYROID ANTAGONISTS E4 **E5** 0 1 THYROID CANCER **E6** 516 3 THYROID CARTILAGE **E7** THYROID CARTILAGE -- ABNORMALITIES -AB **E8** 59 THYROID CARTILAGE -ANATOMY AND **HISTOLOGY AH E9** 4 THYROID CARTILAGE -- BLOOD SUPPLY --BS E10 THYROID CARTILAGE -- CHEMISTRY -- CH E11 3 THYROID CARTILAGE --CYTOLOGY --CY E12 1 THYROID CARTILAGE -- EMBRYOLOGY --EM 5 THYROID CARTILAGE -- GROWTH AND E13 **DEVELOPMENT --G** E14 46 THYROID CARTILAGE --INJURIES --IN E15 2 THYROID CARTILAGE -- INNERVATION -- IR E16 3 THYROID CARTILAGE -- METABOLISM -- ME E17 69 THYROID CARTILAGE --PATHOLOGY --PA E18 25 THYROID CARTILAGE -- PHYSIOLOGY -- PH 8 THYROID CARTILAGE --E19 PHYSIOPATHOLOGY --PP E20 1 THYROID CARTILAGE -- RADIATION **EFFECTS --RE** E21 49 THYROID CARTILAGE -- RADIOGRAPHY --RA **E22** 5 THYROID CARTILAGE -- RADIONUCLIDE IMAGING -- RI 271 THYROID CARTILAGE -SURGERY -SU E24 **6 THYROID CARTILAGE --TRANSPLANTATION** -TR THYROID CARTILAGE --**E25 ULTRASONOGRAPHY --US** E26 4 THYROID CARTILAGE --**ULTRASTRUCTURE --UL** 494 5 THYROID CRISIS **E27 E28** 29 THYROID CRISIS --BLOOD --BL **E29** 30 THYROID CRISIS -- CHEMICALLY INDUCED --CI E30 **THYROID CRISIS -- COMPLICATIONS --**77 CO E31 98 THYROID CRISIS -- DIAGNOSIS -- DI E32 136 THYROID CRISIS -- DRUG THERAPY -- DT E33 2 THYROID CRISIS -- ENZYMOLOGY -- EN 6 E34 THYROID CRISIS -- EPIDEMIOLOGY -- EP E35 123 THYROID CRISIS -- ETIOLOGY -- ET THYROID CRISIS --IMMUNOLOGY --IM E36 3 E37 13 THYROID CRISIS -- METABOLISM -- ME E38 7 THYROID CRISIS -- MORTALITY -- MO E39 12 THYROID CRISIS -- NURSING -- NU E40 5 THYROID CRISIS -- PATHOLOGY -- PA E41 36 THYROID CRISIS -- PHYSIOPATHOLOGY -.pp E42 35 THYROID CRISIS -- PREVENTION AND CONTROL --PC E43 THYROID CRISIS -- RADIOGRAPHY -- RA E44 3 THYROID CRISIS -- RADIONUCLIDE IMAGING --RI **E45** THYROID CRISIS -- RADIOTHERAPY -- RT E46 12 THYROID CRISIS --SURGERY --SU THYROID CRISIS -THERAPY --TH E47 140 E48 9605 21 THYROID DISEASES

192 N 6 EUTHYROID SICK SYNDROMES R5 11460 N 6 GOITER R6 1512 N 2 GOITER, ENDEMIC 2024 N 3 GOITER, NODULAR R7 471 N 3 GOITER, SUBSTERNAL R9 8883 N 15 GRAVES' DISEASE R10 18660 N 6 HYPERTHYROIDISM R11 276 N 3 HYPERTHYROXINEMIA R12 19237 N 5 HYPOTHYROIDISM 9605 DC="C19.874." 786 "CRETINISM" 192 "EUTHYROID SICK SYNDROMES" 11460 "GOITER" 1512 "GOITER, ENDEMIC" 2024 "GOITER, NODULAR" 471 "GOITER, SUBSTERNAL" 8883 "GRAVES' DISEASE" 18660 "HYPERTHYROIDISM" 276 "HYPERTHYROXINEMIA" 19237 "HYPOTHYROIDISM"

111387 S1 OR (S2-S13)

16877 CLOSTRIDIUM

49003 NEUROTOXIN OR TOXIN

14 S14 AND S15

323 S14 AND S17

S3

S4

S5

S6

S7

S8

S9

S10

S11

S12

S13

S14

S15

S16

S17

S18

Items RT Index-term Ref **E1** 34 **BOTULINUM TOXIN TYPE E** F2 **BOTULINUM TOXIN TYPE F** 14 **E3** 3071 11 *BOTULINUM TOXINS F4 568 **BOTULINUM TOXINS --ADMINISTRATION AND DOSAGE -**251 **BOTULINUM TOXINS --ADVERSE** E5 **EFFECTS -- AE BOTULINUM TOXINS --ANALYSIS --AN** 248 **E6 F7** 34 **BOTULINUM TOXINS --ANTAGONISTS AND INHIBITORS BOTULINUM TOXINS --BIOSYNTHESIS** E8 168 -BI **BOTULINUM TOXINS -- BLOOD -- BL** E9 64 E10 **BOTULINUM TOXINS --CHEMICAL** SYNTHESIS -- CS E11 119 **BOTULINUM TOXINS --CHEMISTRY --**CH E12 30 **BOTULINUM TOXINS --CLASSIFICATION --CL**

Ref Items Type RT Index-term 11 *BOTULINUM TOXINS R2 3071 X DC=D24.185.926.123.179. (BOTULINUM TOXINS) R3 3071 X DC=D24.185.926.640.75. (BOTULINUM TOXINS) R4 114 X 1 BOTULIN 0 X 1 CLOSTRIDIUM BOTULINUM TOXINS R5 R6 1785 R 10 BOTULISM R7 628 R 108 CHOLINERGIC AGENTS R8 1519 R 5 CLOSTRIDIUM BOTULINUM R9 301 B 29 ANTI-DYSKINESIA AGENTS R10 9006 B 16 BACTERIAL TOXINS R11 7222 B 15 NEUROTOXINS R12 585 N 4 BOTULINUM TOXIN TYPE A

S19 3071 "BOTULINUM TOXINS" S20 16 S19 AND S14 **S21** 9605 "THYROID DISEASES" S22 0 DELETE S21

S23 0 S3 AND S18 AND (PERFRINGENS) S24

8 S21 AND S

16/6/1 10026619 99097345 PMID: 9878248 Genes for the CPE receptor (CPETR1) and the human homolog of RVP1(CPETR2) are localized within the Williams-Beuren syndrome deletion. Dec 15 1998

16/6/2 04722792 81133651 PMID: 6258596 Demonstration and characterization of partial glyceride specific lipases in pig thyroid plasma membranes. Nov 28 1980

16/6/3 04661467 84263155 PMID: 6378767

regulation of the immune response to tetanus toxoid in o's disease. Jul 1984

16/6/4 04201303 81164223 PMID: 7215238 Gas-forming suppurative thyroiditis. Mar 1981

16/6/5 02675218 77138183 PMID: 191247 Effects of concanavalin A and neuraminidase on cyclic AMP levels and 14C-1-glucose oxidation in dog thyroid slices. Aug 1976

16/6/6 02255653 69254786 PMID: 4895160 [Various findings on the processes of natural detoxication of the body during infectious processes] Nekotorye dannye o protsessakh estestvennoi detoksikatsii organizma pri infektsionnykh protsessakh.Oct

16/6/7 02092271 74150094 PMID: 4363284 Possiblity of cancer diagnosis by detection of Clostridial antibodies. 1972

16/6/8 02044882 71067137 PMID: 4321500 Endemic goiter in Greece: some new epidemiologic studies. Feb 1971

16/6/9 01943289 73001076 PMID: 4560621 Treatment of malignant tumors with spores of Clostridium butyricum M 55. 3. Therapeutic experiments on metastasizing tumors of various organs] Die Behandlung matigner Geschwulste mit sporen von Clostridium butyricum M 55. 3. Therapieversuche an metastasierenden Geschwulsten ver schiedener Organe. 1972

16/6/10 01695705 67139798 PMID: 4960830 Action of phospholipase C on the thyroid. Abolition of the response to thyroid-stimulating hormone. Apr 25 1967

16/6/11 01512434 70106909 PMID: 4312983 Role of lecithin in the mechanism of TSH action. Apr 1970

16/6/12 01502036 68318489 PMID: 4298230 Effect of sphingomyelinase from Clostridium perfringens on the metabolic activity and phospholipid composition of thyroid slices. Jul 1

16/6/13 01496873 67221632 PMID: 4292226 The purification and properties of a thyroid-stimulating factor isofated from Clostridium perfringens. Aug 25 1967

16/6/14 00882933 71059144 PMID: 5487765 Clostridium septicum infection of the thyroid gland. Sep 1970

90754 "THYROID" **S1** S2 9605 "THYROID DISEASES"

R1

Ref Items Type RT Index-term

9605 21 *THYROID DISEASES 9605 X DC=C19.874. (THYROID DISEASES) R3 786 N 9 CRETINISM

16/7/1 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Serporation. All rts. reserv. 10026619 99097345 PMID: 9878248

Genes for the CPE receptor (CPETR1) and the human homolog of RVP1 (CPETR2) are localized within the Williams-Beuren syndrome deletion.

Paperna T; Peoples R; Wang YK; Kaplan P; Francke U

Department of Genetics, Stanford University School of Medicine, Stanford, California, 94305, USA.

Genomics (UNITED STATES) Dec 15 1998, 54 (3) p453-9, ISSN 0888-7543 Journal Code: GEN Contract/Grant No.: HD01181, HD, NICHD; HD33505, HD, NICHD; HG00298, HG, NHGRI Languages: ENGLISH Document type: Journal Article Record type: Completed

Williams-Beuren syndrome (WBS) is a neurodevelopmental disorder affecting multiple systems. Haploinsufficiency of genes deleted in chromosomal region 7q11.23 is the likely cause for this syndrome. We now report the localization of the genes for the CPE-R (Clostridium perfringens enterotoxin receptor, CPETR1) and the human homolog of RVP1 (rat ventral prostate 1 protein, CPETR2), both previously mapped to 7q11, to the WBS critical region. A single nucleotide polymorphism (SNP) present in CPETR1 has been identified and was used to determine parental ongin of the deleted allele in five informative families. The mouse homologs Cpetr1 and Cpetr2 were identified and mapped to the conserved syntenic region on mouse chromosome 5. Northerm blot analysis of CPETR1 demonstrates tissue specificity, with expression in kidney, lung, thyroid, and gastrointestinal tissues. In mouse, Cpetr1 is expressed in the early embryo, appears to be developmentally upregulated during gestation, and is present in adult tissues. Our results suggest a role for CPE-R in internal organ development and function during pre- and postnatal life. Copyright 1998 Academic Press. Record Date Created: 19990223

16/7/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04722792 81133651 PMID: 6258596

Demonstration and characterization of partial glycende specific lipases in pig thyroid plasma membranes.

Igarashi Y: Kondo Y

Biochemical and biophysical research communications (UNITED STATES) Nov 28 1980, 97 (2) p766-71, ISSN 0006-291X Journal Code: 9Y8 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19810413

16/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04661467 84263155 PMID: 6378767

Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease. Fawcett J; Hutton C; Mclachlan SM; Clark F; Rees Smith B

Immunology (ENGLAND) Jul 1984, 52 (3) p525-8, ISSN 0019-2805 Journal Code: GH7 Languages: ENGLISH Document type: Journal Article Record type: Completed The humoral immune response to tetanus toxoid has been studied in patients with Hashimoto's disease. Although the magnitude of the response was similar to that observed in normal subjects, the Hashimoto patients demonstrated an inability to regulate their levels of tetanus toxoid antibody. This apparent defect in the control of antibody synthesis may be an important factor in both the initiation and perpetuation of autoimmune thyroid disease. Record

16/7/4 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04201303 81164223 PMID: 7215238

Gas-forming suppurative thyroiditis.

Date Created: 19840829

Michel RG; Hall DM; Woodard BH

Ear, nose, & throat journal (UNITED STATES) Mar 1981, 60 (3) p127-30, ISSN 0145-5613 Journal Code: EDF

Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19810613

16/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 02675218 77138183 PMID: 191247

18/6/1 11223191 21167934 PMID: 11267996

Mechanisms of P2 receptor-evoked DNA synthesis in thyroid FRTL-5 cells. May 2001

18/6/2 10931041 20494849 PMID: 11041451

The thyrotropin receptor is not involved in the activation of p42/p44 mitogen-activated protein kinases by thyrotropin preparations in Chinese hamster ovary cells expressing the human thyrotropin receptor. Sep 2000

18/6/3 10880922 20406492 PMID: 10951975

Altered expression of G proteins in thyroid gland adenomas obtained from hyperthyroid cats. Aug 2000

18/6/4 10709815 20327831 PMID: 10867750 Is autism a G-alpha protein defect reversible with natural vitamin A? Jun 2000

18/6/5 10648056 20322683 PMID: 10866314

Cancer gene-therapy by thyroid hormone-mediated expression of toxin genes. Jun 15 2000

18/6/6 10607495 20252140 PMID: 10794166 Study of the olivocochlear neurons using two different tracers, fast blue and cholera toxin, in hypothyroid rats. Apr 2000

18/6/7 10557836 20202319 PMID: 10737891

Extracellular ATP-mediated phospholipase A(2) activation in rat thyroid FRTL-5 cells: regulation by a G(i)/G(o) protein, Ca(2+), and mitogenactivated protein kinase. May 2000

18/6/8 10516902 20163705 PMID: 10701770 Effect of endotoxin challenge on hepatic 5'-deiodinase activity in cattle. Jan 2000

18/6/9 10513968 20115224 PMID: 10648115 Adjuvant effects of cholera toxin b subunit on immune response to recombinant thyrotropin receptor in mice. Feb 2000

18/6/10 10359010 20000371 PMID: 10532571 What is the role of botulinum toxin in the treatment of dysthyroid strabismus? Oct 1999

18/6/11 10358931 20000569 PMID: 10532769 Strabismus surgery among aged medicare beneficiaries. Dec 1997

18/6/12 10341274 99328094 PMID: 10401667
An adenosine receptor agonist-induced modulation of TSH-dependent cell growth in FRTL-5 thyroid cells mediated by inhibitory G protein, Gi. Apr 1999

18/6/13 10336103 99262181 PMID: 10329469

Effects of concanavalin A and ne pidase on cyclic AMP levels and 14C-1-glucose oxidation in dog thyroid slices.

Yamashita K; Aiyoshi Y; Oka H; Ogata E

Endocrinologia japonica (JAPAN) Aug 1976, 23 (4) p355-8, ISSN 0013-7219 Journal Code: EG5 Languages: ENGLISH Document type: Journal Article Record type: Completed Treatment with concanavalin A at 100 micron/ml or higher concentrations significantly increased 14C-1-glucose oxidation in dog thyroid slices as reported in other tissues. This treatment exerted no effect on tissue cyclic AMP levels. Neuraminidase at the same concentrations also had similar effects on these parameters. Neither concanavalin A nor neuraminidase at the concentrations up to 100 microng/ml had the TSH effect on both tissue cyclic AMP and 14C-1-glucose oxidation. These results indicate that modification of carbohydrate moieties of glycoproteins on the cell surface may cause an increase in glucose metabolism without any critical effect on cyclic AMP system and in the process of TSH response. Record Date Created: 19770527

16/7/9 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 01943289 73001076 PMID: 4560621

Treatment of malignant tumors with spores of Clostridium butyricum M 55. 3. Therapeutic experiments on metastasizing tumors of various organs] Die Behandlung maligner Geschwulste mit sporen von Clostridium butyricum M 55. 3. Therapieversuche an metastasierenden Geschwulsten ver schiedener Organe.

Kretschmer H; Glaser A; Grasser A
Archiv fur Geschwulstforschung (GERMANY, EAST) 1972, 39 (4) p315-21, ISSN 0003-911X
Journal Cod e: 746 Languages: GERMAN Document type: Clinical Trial; Journal Article
Record type: Completed Record Date Created: 19721110

16/5/10 DIALOG(R)File 155:MEDLINE(R) (c) formationly 2001 Dialog Corporation. All rts. reserv. 01695705 67139798 PMID: 4960830

Action of phospholipase C on the thyroid. Abolition of the response to thyroid-stimulating hormone.

Macchia V; Pastan I

Journal of biological chemistry (UNITED STATES) Apr 25 1967, 242 (8) p1864-9, ISSN 0021-9258 Journal Code: HIV Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19670706

Tags: Animal; In Vitro

Descriptors: Phospholipases—pharmacology—PD; *Thyroid Gland—drug effects—DE; *Thyrotropin—pharmacology—PD; Acetylcholine—pharmacology—PD; Clostridium —enzymology—EN; Dogs; Edetic Acid—pharmacology—PD; Glucose-metabolism—ME; Neuraminidase—metabolism—ME CAS Registry No.: 50-99-7 (Glucose); 51-84-3 (Acetylcholine); 60-00-4 (Edetic Acid); 9002-71-5

Enzyme No.: EC 3.1.- (Phospholipases); EC 3.2.1.18 (Neuraminidase)

16/5/13 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 01496873 67221632 PMID: 4292226

The purification and properties of a thyroid-stimulating factor isolated from Clostridium perfringens.

Macchia V; Bates RW; Pastan I

Journal of biological chemistry (UNITED STATES) Aug 25 1967, 242 (16) p3726-30, ISSN 0021-9258 Journal Code: HIV Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19671022

Tags: Animal; In Vitro

Descriptors: Bacterial Proteins—analysis—AN; *Bacterial Proteins—pharmacology—PD; *Clostridium perfringens—analysis—AN; *Thyroid Gland-metabolism—ME; Carbon Dioxide; Carbon Isotopes; Cattle; Chromatography, Gel; Dogs; Glucose-metabolism—ME; Molecular Weight; Peptide Hydrolases; Phosphates—metabolism—ME; Phospholipids—biosynthesis—BI; Thyrotropin; Trypsin

Cattle; Chromatography, Get, Doys, Choose-instabulish-mis, Innaectical Trogging Spaces (Phosphates-metabolism-ME; Phospholipids-biosynthesis-Bl; Thyrotropin; Trypsin CAS Registry No.: 0 (Bacterial Proteins); 0 (Carbon Isotopes); 0 (Phospholipids); 124-38-9 (Carbon Dioxide); 50-99-7 (Clucose); 9002-71-5 (Thyrotropin)
Enzyme No.: EC 3.4 (Peptide Hydrolases); EC 3.4.21.4 (Trypsin)

kinase in cultured cells. May 1999

Sphingosylphosphorylcholine activates Gq, Gi-2, and Gi-3 in thyroid FRTL-5 cells: implications for the activation of calcium fluxes and Na++ exchange. May 19 1999

18/6/14 10335016 99261834 PMID: 10329948
Thyroid hormone induces activation of mitogen-activated protein

18/6/15 10291747 97423434 PMID: 9277376 Effects of triiodothyronine administration on the adenylyl cyclase system in brown adipose tissue of rat. Aug 1997

18/6/16 10240117 99371424 PMID: 10443824 The posterior thyroplasty window: anatomical considerations. Aug 1999

18/6/17 10180055 99302523 PMID: 10374293 [Systemic manifestations of myasthenia gravis and its putative pathogenesis] Jun 1997—

18/6/18 10076868 99196145 PMID: 10098509
Thyrotropin regulates c-Jun N-terminal kinase (JNK) activity through two distinct signal pathways in human thyroid cells. Apr 1999

18/6/19 10058220 99165190 PMID: 10067867 Regulation and transfer of a murine model of thyrotropin receptor antibody mediated Graves' disease. Mar 1999 18/6/20 10021711 99082623 PMID: 9865104

Bilateral laryngeal movement disorder and synkinesia: value of botulism toxin. Apropos of a case] Trouble de la mobilite laryngee bilaterale et syncinesies: interet de la toxine botulique. A propos d'un cas. 1998

18/6/21 09880412 98421840 PMID: 9751220

Cyclic AMP impairs the PRL stimulation of iodide uptake into mouse mammary tissues. Oct 1998

18/6/22 09828189 98361458 PMID: 9697993

Characterization of the murine immune response to the murine TSH receptor ectodomain: induction of hypothyroidism and TSH receptor antibodies. Jul 1998

18/6/23 09825762 98378683 PMID: 9713061

Botulinum toxin A treatment of overactive corrugator supercilii in thyroid eye disease. May 1998

18/6/24 09783338 98283956 PMID: 9618427

Effect of antithyroid drugs on hydroxyl radical formation and alpha-1-proteinase inhibitor inactivation by neutrophils: therapeutic implications. Jun 1998

18/6/25 09700286 98184392 PMID: 9525480

Protein tyrosine phosphorylation and calcium signaling in thyroid FRTL-5 cells. May 1998

18/6/26 09674340 98070720 PMID: 9405207

Loss of biological activity due to Glu-->Arg mutation at residue 11 of the B subunit of cholera toxin. Nov 1997

18/6/27 09589335 97462684 PMID: 9322911

Sphingosine 1-phosphate mobilizes sequestered calcium, activates calcium entry, and stimulates deoxyribonucleic acid synthesis in thyroid FRTL-5 cells. Oct 1997

18/6/28 09579710 97424751 PMID: 9278864

Somatostatin blocks the potentiation of TRH-induced TSH secretion from perifused pituitary fragments and the change in intracellular calcium concentrations from dispersed pituitary cells elicited by prepro-TRH (PS4) or by tri-iodothyronine. Aug 1997

18/6/29 09568457 97405869 PMID: 9260913

The phosphatase inhibitor okadaic acid stimulates the TSH-induced G1-S phase transition in thyroid cells. Aug 1 1997

18/6/30 09470883 98025576 PMID: 9376224

Sodium saccharin inhibits adenylyl cyclase activity in non-taste cells. Sep 1997

18/6/31 09469678 97375443 PMID: 9231760

Thyroid specific expression of cholera toxin A1 subunit causes thyroid hyperplasia and hyperthyroidism in transgenic mice. Aug 1997

18/6/32 09463997 97254493 PMID: 9099903

Multiple G-protein coupling of the dog thyrotropin receptor. Mar 14 1997

18/6/33 09459986 97240583 PMID: 9124507 Sphingosine 1-phosphate stimulates Na+/H+ exchange in thyroid

FRTL-5 cells. Mar 1997

18/6/34 09455989 97131950 PMID: 8977407

Sphingosine 1-phosphate stimulates hydrogen peroxide generation through activation of phospholipase C-Ca2+ system in FRTL-5 thyroid cells: possible involvement of guanosine triphosphate-binding proteins in the lipid signaling. Jan 1997

18/6/35 09428059 98018536 PMID: 9380360 Chemodeneryation in treatment of unper eyelid retraction 19

Chemodenervation in treatment of upper eyelid retraction. 1997

18/6/36 09408392 98018742 PMID: 9376076 TCR vbeta usage of TSH receptor-specific CD4+ T cells in Graves'

disease patients and healthy humans. Oct 1997

18/6/37 09372529 97366554 PMID: 9223384

N6-isopentenyladenosine affects cAMP-dependent microfilament

organization in FRTL-5 thyroid cells. Jul 10 1997 18/6/38 09342718 97329812 PMID: 9186271

Assessment of thyroid growth stimulating activity of immunoglobulins from patients with autoimmune thyroid disease by cytokinesis arrest assay. May 1997

18/6/39 09338863 97324798 PMID: 9180903

Adenosine inhibits DNA synthesis stimulated with TSH, insulin, and phorbol 12-myristate 13-acetate in rat thyroid FRTL-5 cells. Jun 1997

18/6/40 09270589 97184183 PMID: 9030595

Basolateral localization and transcytosis of gonadotropin and thyrotropin receptors expressed in Madin-Darby canine kidney cells. Feb 21 1997

18/6/41 09245241 97094291 PMID: 8940379

Effects of hypothyroidism on brown adipose tissue adenylyl cyclase activity. Dec 1996

09245237 97094274 PMID: 8940362

Transformation of rat thyroid follicular cells stably transfected with cholera toxin A1 fragment. Dec 1996

18/6/43 09229599 97046665 PMID: 8891586

Amitriptyline inhibits the G protein and K+ channel in the cloned thyroid cell line. Sep 19 1996

18/6/44 09224622 96290945 PMID: 8754735

Increased cyclic adenosine 3',5'-monophosphate inhibits G proteincoupled activation of phospholipase C in rat FRTL-5 thyroid cells. Aug 1996

18/6/45 09159599 97154483 PMID: 9001201 Thyroid hormones as neurotransmitters. Dec 1996

18/6/46 09076947 97110552 PMID: 8952703

Puriner gic agonists stimulate the secretion of endothelin-1 in rat thyroid FRTL-5 cells. Dec 1996

18/6/47 09036686 96290969 PMID: 8754759

Thyrotropin (TSH) receptor antibodies (TSHrAb) can inhibit TSHmediated cyclic adenosine 3',5'- monophosphate production in thyroid cells by either blocking TSH binding or affecting a step subsequent to TSH binding. Aug 1996

18/6/48 09036613 97050599 PMID: 8895327

Transfer of thyroiditis, with syngeneic spleen cells sensitized with the human thyrotropin receptor, to naive BALB/c and NOD mice. Nov 1996

18/6/49 09028997 96426631 PMID: 8828910

Inhibitions of protein kinases and protein phosphatases have opposite effects on thyrotropin-stimulated cAMP accumulation in human thyroid cells. Jun 1996

18/6/50 09005228 96328895 PMID: 8735590

An ade nosine derivative cooperates with TSH and Graves' IgG to induceCa2+ mobilization in single human thyroid cells. Apr 19 1996

18/6/51 08916239 96217296 PMID: 8641209

Effect of thyroid hormones on G proteins in synaptosomes of chick embryo. Jun 1996

18/6/52 08910353 96159051 PMID: 8591983

Purinergic agonist ATP is a comitogen in thyroid FRTL-5 cells. Feb 1996

18/6/53 08909827 96133888 PMID: 8552586

The human thyrotropin receptor: a heptahelical receptor capable of stimulating members of all four G protein families. Jan 9 1996

18/6/54 08896009 95280650 PMID: 7760657

Growth and invasion of differentiated thyroid gland carcinoma: importance of signal transduction] Wachsturn und Invasion beim differenzierten Schilddrusenkarzinom: Stellenwert der Signaltransduktion. 1995

18/6/55 08895568 95271486 PMID: 7752073

Antiarrhythmic drugs inhibit the G-protein and K+ channels in the cultured thyroid cell line. May 1995

18/6/56 08894783 95250934 PMID: 7733252

Mechan isms of action of somatostatin on human TSH-secreting adenoma cells. Apr 1995

18/6/57 08893052 95188900 PMID: 7882998

Stimulation of mitogen-activated protein kinase by thyrotropin in astrocytes. Feb 15 1995 $\,$

18/6/58 08892884 95181324 PMID: 7876108

Stimulation of mitogen-activated protein kinase by thyrotropin in primary cultured human thyroid follicles. Feb 24 1995

18/6/59 08891996 95129470 PMID: 7828520

Thyrotr opin-induced hydrogen peroxide production in FRTL-5 thyroid cells is mediated not by adenosine 3',5'-monophosphate, but by Ca2+ signaling followed by phospholipase-A2 activation and potentiated by an adenosine derivative. Jan 1995

18/6/60 08886263 95076361 PMID: 7985086

An in vitro model of thyroid neoplasia: permanently transfected FRTL-5 cells with thyroglobulin promoter-cholera toxin A1 subunit minigene. Dec 1994

18/6/61 08883992 95025566 PMID: 7939340

[G-proteins and endocrine tumors. The example of acromegaly] Proteines G et tumeurs endocrines. L'exemple de l'acromegalie. May 1 1994

18/6/62 08860246 94351732 PMID: 7915334

Enhanced negative inotropic effect of an adenosine A1-receptor agonist in rat left atria in hypothyroidism. Apr 1994

18/6/63 08858318 94307166 PMID: 8033808

1,25 Dihydroxyvitamin D3 attenuates adenylyl cyclase activity in rat hy lls: reduction of thyrotropin receptor number and increase in guaranteelide-binding protein Gi-2 alpha. Aug 1994

18/6/64 08847202 96198112 PMID: 8626445

Acidification of serotonin-containing secretory vesicles induced by a plasma membrane calcium receptor. Mar 15 1996

18/6/65 08806554 96121532 PMID: 8557239

Adenosine A1-receptors inhibit cAMP and Ca2+ mediated calcitonin secretion in C-cells. Sep 1995

18/6/66 08754639 96368096 PMID: 8772249

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Tags: Case Report; Female; Human; Male; Support, Non-U.S. Gov't Descriptors: Botulinum Toxins—therapeutic use--TU; "Ophthalmoplegia—drug therapy—DT; "Thyroid Diseases—complications—CO; Adult; Aged; Amblyopia—drug therapy—DT; Amblyopia—etiology—ET; Botulinum Toxins—adverse effects—AE; Middle Age; Ophthalmoplegia—complications—CO; Ophthalmoplegia—

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18/7/10 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 10359010 20000371 PMID: 10532571

What is the role of botulinum toxin in the treatment of dysthyroid strabismus?

Gair EJ; Lee JP; Khoo BK; Maurino V

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Journal of AAPOS (UNITED STATES) Oct 1999, 3 (5) p272-4, ISSN 1091-8531 Journal Code: C99 Languages: ENGLISH Document type: Journal Article Record type: Completed BACKGROUND: Botulinum toxin A has been used in the treatment of dysthyroid strabismus primarily as a temporary measure during the active phase of the disease. We report on our experience with 65 patients. METHOD: We review the records of 65 patients with dysthyroid strabismus who were treated with botulinum toxin A at Moorfields Eye Hospital between 1984 and

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1996. CONCLUSIONS: Patients with a short duration of relatively mild dysthyroid strabismus

A in cases of severe dysthyroid disease. Record Date Created: 19991123

have a chance of long-term benefit with botulinum toxin A. There is little use for botulinum toxin

Strabismus surgery among aged medicare beneficiaries.

Repka MX

Wilmer Ophthalmological Institute, Johns Hopkins Hospital, Baltimore, MD 21287-9009, USA. Journal of AAPOS (UNITED STATES) Dec 1997, 1 (4) p231-4, ISSN 1091-8531 Journal Code: C99 Languages: ENGLISH Document type: Journal Article Record type: Completed

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OBJECTIVES: The purpose of this study was to investigate the inc of strabismus surgery among aged patients in the United States. METHODS: The Medicare Fart B claims experience (physician professional fee billing) for 1995 was reviewed for the number of times each strabismus surgical procedure recognized in Physicians' Current Procedural Terminology (CPT) was performed. To determine the indications for the procedures that were performed, a 5% sample of claims was reviewed for the pertinent International Classification of Diseases, Ninth Revision, Clinical Modification, diagnostic codes, RESULTS: There were 27 million aged Medicare beneficiaries eligible for Part B benefits in 1995 in a fee-for-service setting. During that year physicians reported 9497 strabismus physician services. These represented 6585 separate procedures (CPT codes 67311 to 67343) and 277 botulinum toxin (Botox) injections for strabismus (CPT 67345) performed during 1995. Sixty-nine percent of the surgical procedures were for horizontal correction and 28% were for vertical correction. Adjustable sutures were used for only 1240 cases (1 9%). The add-on procedural code for reoperation surgery or surgery in the presence of restriction of the extraocular muscles was used in just 930 cases (14%). The most common diagnosis for honzontal surgery was exotropia. Paralytic strabismus and thyroid disease were identified for 17% of cases. Three percent of the diagnoses were inappropriate for the procedures performed and may have been reported in error. CONCLUSIONS: These data confirm a very low incidence of strabismus surgical procedures (2/10,000) and injections (1/100,000) among aged Medicare beneficiaries. The strabismus surgery was most often performed to repair a horizontal deviation. The adjustable suture technique was used infrequently. These data may be extrapolated into the future to aid in determining the strabismus services that will be needed early in the next century. Record Date Created: 19991102

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Bilateral laryngeal movement disorder and synkinesia: value of botulism toxin. Apropos of a case] Trouble de la mobilite laryngee bilaterale et syncinesies: interet de la toxine botulique. A propos d'un cas.

Marie JP; Navarre I; Lerosey Y; Magnier P; Dehesdin D; Andrieu Guitrancourt J C.H.U. Rouen, Service d'ORL et Chirurgie Cervico-Faciale, France.

Revue de laryngologie - otologie - rhinologie (FRANCE) 1998, 119 (4) p261-4, ISSN 0035-1334 Journal Code: SDD

Languages: FRENCH Document type: Journal Article Record type: Completed Several years after a subtotal thyroidectomy complicated by bilateral vocal cord paralysis, the patient presented with progressive dyspnea due to laryngeal synkinesis. The impairement of the ventilation status, in spite of laser arytenoidectomy, followed by contralateral posterior transverse cordotomy, suggested a botulinum toxin injection in the intrinsic adductor laryngeal muscles. The rapid improvement in ventilation without phonatory impairement is discussed in the following report. Record Date Created: 19990212

18/7/23 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09825762 98378683 PMID: 9713061

Botulinum toxin A treatment of overactive corrugator supercilii in thyroid eye disease. Olver JM

Western Eye Hospital, London.

British journal of ophthalmology (ENGLAND) May 1998, 82 (5) p528-33, ISSN 0007-1161 Journal Code: AZK Languages: ENGLISH Document type: Clinical Trial; Journal Article Record type: Completed

BACKGROUND/AIM: Patients with thyroid eye disease with upper eyelid retraction often develop overaction of the accessory muscles of eyelid closure, the glabellar muscles corrugator supercilii and procerus. The resultant glabellar furrowing (frown lines) contributes to the typical thyroid facies. The aim of this study was to evaluate the use of botulinum toxin A reversible chemodenervation of the glabellar muscles as adjunctive treatment in the rehabilitation of patients with thyroid eye disease. METHODS: 14 patients (13 females) ages 39-76 years (mean 52) with inactive thyroid eye disease and associated medial eyebrow ptosis and prominent glabellar frown lines were recruited. All patients had a history of upper eyelid retraction. Each patient was treated with a single botulinum toxin injection (Dysport 0.2 ml, 40 units) into each corrugator supercilii and sometimes procerus muscles as an outpatient procedure. The effectiveness and acceptability of the treatment was assessed clinically and from a patient questionnaire. RESULTS: The injections were tolerated by 13/14 (93%) patients. There was resultant flattening of the glabellar region and improvement of medial eyebrow contour in all patients, with onset of paralysis within 1 week. All patients reported a subjective improvement in appearance. Side effects included one patient (7%) with reversible partial ptosis. The beneficial effect lasted 4-6 months, with a gradual return of function. Repeat treatment was indicated where there was persistent upper eyelid retraction and protractor overaction. CONCLUSION: Botulinum toxin. A chemodenervation of the glabellar muscles in these patients was effective and acceptable. Chemodenervation should be considered in the rehabilitation of patients with thyroid eye disease where there is upper eyelid retraction and overacting protractors resulting in a thyroid frown. Once the eyelid retraction has been successfully treated by surgery, the need for further glabella muscle chemodenervation is considerably reduced. Record Date Created: 19980827

18/7/24 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09783338 98283956 PMID: 9618427

Effect_of_antithyroid_drugs_on-hydroxyl-radical_formation-and_alpha-1-proteinase—inhibitor inactivation_by_neutrophils: therapeutic implications.

Ross AD; Dey I; Janes N; Israel Y

Department of Pharmacology, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada M5S 1A8.

Journal of pharmacology and experimental therapeutics (UNITED STATES) Jun 1998, 285 (3) p1233-8, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: 5P50 AA07186, AA, NIAAA; R01 AA10967, AA, NIAAA; R37-AA10967, AA, NIAAA

Languages: ENGLISH Document type: Journal Article Record type: Completed

The release of proteolytic enzymes eneration of strong oxidants such as the hydroxyl radical by activated neutrophils has roposed to play an important role in mediating toxin induced liver injury. The antithyroid drug propylthiouracil protects against liver injury induced by many hepatotoxic agents and markedly reduces mortality in patients with alcoholic liver disease. However, the mechanism(s) by which propylthiouracil protects against liver injury is not well understood. The present studies investigate the effect of antithyroid drugs on proteolytic enzyme activity and on hydroxyl radical generation from activated neutrophils. In the presence of hydrogen peroxide and chloride, neutrophil myeloperoxidase, an enzyme from the same gene superfamily as thyroid peroxidase, generates hypochlorous acid which inactivates alpha-1proteinase inhibitor (A1PI) present in serum. This inactivation allows neutrophil-released proteolytic enzymes to attack cells. In the present study myeloperoxidase activity was inhibited fully at therapeutic concentrations by antithyroid drugs (propylthiouracil and methimazole). Antithyroid drugs fully prevented hypochlorous acid formation, and prevented neutrophil-mediated inactivation of A1PI, with concomitant blockage of proteolytic activity. Conversely, generation of both superoxide and hydroxyl radicals by activated neutrophils was unaffected by propylthiouracil. The production of these oxygen radicals was fully inhibited by the NADPH oxidase inhibitor diphenylene iodonium chloride, however. These studies indicate that antithyroid drugs are unlikely to prevent cell injury by inhibiting hydroxyl radical generation or by scavenging hydroxyl radicals, but are likely to exert their hepatoprotective anti-inflammatory action by inhibiting neutrophil myeloperoxidase, an enzyme akin to thyroid peroxidase. Record Date Created: 19980702

18/7/25 DIALOG(R)File 155:MEDLINE(R) (c) formationly 2001 Dialog Corporation, All rts. reserv. 09700286 98184392 PMID: 9525480

Protein tyrosine phosphorylation and calcium signaling in thyroid FRTL-5 cells.

Tomquist K; Dugue B; Ekokoski E

Department of Biosciences, University of Helsinki, Finland.

Journal of cellular physiology (UNITED STATES) May 1998, 175 (2) p211-9, ISSN 0021-9541 Journal Code: HNB Languages: ENGLISH Document type: Journal Article Record type: Completed

We examined the importance of tyrosine kinase(s) on the ATP-evoked Ca2+ entry and DNA synthesis of thyroid FRTL-5 cells. ATP rapidly and transiently tyrosine phosphorylated a 72-kDa protein(s). This phosphorylation was abolished by pertussis toxin and by the tyrosine kinase inhibitor genistein, and was dependent on Ca2+ entry. Pretreatment of the cells with genistein did not affect the release of sequestered Ca2+, but the capacitative Ca2+ or Ba2+ entry evoked by ATP or thapsigargin was attenuated. Pretreatment of the cells with orthovanadate enhanced the increase in intracellular free Ca2+ ([Ca2+ji), whereas the Ba2+ entry was not increased. Phorbol 12-mynistate 13-acetate (PMA) phosphorylated the same protein(s) as did ATP. Genistein inhibited the ATP-evoked phosphorylation of MAP kinase and attenuated both the ATP- and the PMA-evoked DNA synthesis. However, genistein did not inhibit the ATP-evoked expression of c-fos. Furthermore, genistein enhanced the ATP-evoked release of arachidonic acid. Thus, ATP activates a tyrosine kinase via a Ca2+-dependent mechanism. A genistein-sensitive mechanism participates, in part, in the ATP-evoked activation of DNA synthesis. Genistein inhibits only modestly capacitative Ca2+ entry in FRTL-5 cells. Record Date Created: 19980416

18/7/26 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09674340 98070720 PMID: 9405207

Loss of biological activity due to Glu--->Arg mutation at residue 11 of the B subunit of cholera toxin.

Yamaoka J; Yamasaki S; Kurazono H; Imamura S; Noda M; Miyai K; Takeda Y Faculty of Medicine, Kyoto University, Kyoto, Sakyo-ku, 606-01, Japan. Microbial pathogenesis (ENGLAND) Nov 1997, 23 (5) p297-302, ISSN 0882-4010 Journal Code: MIC

Languages: ENGLISH Document type: Journal Article Record type: Completed Since it has been reported that a single amino acid mutation of Gly—Arg in the CAGYC region of the beta chain of human thyroid stimulating hormone (hTSH) was responsible for congenital isolated TSH deficiency, and that the same amino acid substitution in this site of hTSH and human chorionic gonadotropin (hCG) introduced by site-directed mutagenesis resulted in loss of activity, the authors studied the role of glutamic acid at position 11 (Glu-11) from the N-terminus of the B subunit of cholera toxin (CT), which corresponds to the glycine in the CAGYC region of the beta chain of hTSH and hCG. A mutant CT constructed by site-directed mutagenesis in which Glu-11 was replaced by Arg (CT-E11R) did not induce either morphological changes or accumulation of cytosolic cyclic AMP in Chinese hamster ovary cells, although it formed the holotoxin AB5, retained the ability to bind to GM1-ganglioside and showed ADP-ribosyltransferase activity. Weak assembly of the B subunits in mutant CT-E11R demonstrated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis under non-heating conditions might explain the loss of biological activity. Copyright 1997 Academic Press Limited. Record Date Created: 19980317

18/7/31 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09469678 97375443 PMID: 9231760

Thyroid -specific expression of cholera toxin A1 subunit causes thyroid hyperplasia and hyperthyroidism in transgenic mice.

Zeiger MA; Saji M; Gusev Y; Westra WH; Takiyama Y; Dooley WC; Kohn LD; Levine MA
Department of Surgery, Johns Hopkins Medical Institutions, Baltimore, Maryland 21205, USA.

Endocrinology (UNITED STATES) Aug 1997, 138 (8) p3133-40, ISSN 0013-7227

Journal Code: EGZ Contract/Grant No.: RO-1 DK34281, DK, NIDDK Languages: ENGLISH
Document type: Journal Article Record type: Completed

Thyroid cell growth and function are regulated by hormones and growth factors binding to cell surface receptors that are coupled via G proteins, Gs and Gq, to the adenylyl cyclase and phospholipase C signal transduction systems, respectively. Activating mutations of the TSH receptor and G alpha s have been documented in subsets of thyroid neoplasms. To test the oncogenic potential of activated G alpha s in transgenic mice, we used the cholera toxin A1 subunit that constitutively activates G alpha s and used the rat thyroglobulin gene promoter for

targeting this transgene (TGCT) to thyroid follicular cells. Three (M1 1358, and F1286) of six founders identified were able to transmit the transgene to their of and thyroid glands from these mice contained elevated levels of cAMP. Concentrations of serum thyroxine were elevated as early as 2 months of age (M 1392 and F 1286). F1358 mice were euthyroid until 8 months of age, at which time they developed hyperthyroidism. All three TGCT lines developed thyroid hyperplasia independent of their thyroxine levels. DNA image analysis of thyroid follicular cells from both the hyper and euthyroid mice showed that DNA index and "S+G2/M" phase were increased compared with normal, changes similar to that seen in poor prognosis human carcinomas. These data suggest that the G alpha s-adenylyl cyclase-cAMP pathway has an important role in thyroid hyperplasia and the transgenic mouse models reported herein will allow further examination of the role of this pathway in thyroid oncogenesis. Record Date Created: 19970815

18/7/38 DIALOG(R)File 155:MEDLINE(R) (c) formationly 2001 Dialog Corporation, All rts. reserv. 09342718 97329812 PMID: 9186271

Assessment of thyroid growth stimulating activity of immunoglobulins from patients with autoimmune thyroid disease by cytokinesis arrest assay.

Miyamoto S; Kasagi K; Alam MS; Misaki T; Iida Y; Konishi J

Department of Nuclear Medicine, Faculty of Medicine, Kyoto University, Japan.

European journal of endocrinology (ENGLAND) May 1997, 136 (5) p499-507, ISSN 0804-4643 Journal Code: BXU Languages: ENGLISH Document type: Journal Article

Record type: Completed

OBJECTIVE: To develop a novel bioassay for the assessment of thyroid cell growth stimulating activity using cytochalasin B (CB) and to test immunoglobulins (IgGs) from patients with autoimmune thyroid diseases. DESIGN: The assay is based on the principle that growing cells during incubation with CB show an increased number of nuclei in a cell (N/C index), since CB, at appropriate concentrations, is known to inhibit cytoplasmic cleavage without affecting nuclear mitosis. The N/C index represents potential DNA production while cells are incubated with CB. METHODS: FRTL-5 thyroid cells were incubated with various thyroid stimulators in TSH-free medium containing 2 mg/J CB for 3 days. After the incubation, the cells were harvested in trypsin/EDTA to obtain single cell suspension, fixed, dropped onto a glass slide, stained and observed under a microscope to determine the N/C index. RESULTS: Bovine TSH at 10(-3)-1.0 U/I, forskolin at 1x10(-7)-10(-5) mol/I, cholera toxin at 10x10(-5)-10(-3) mg/I, or (Bu)2cAMP at 1 x 10(-5)-10(-3) mol/l increased the N/C index up to approximately 2.0 in a dose-dependent manner. IgGs not only from 27 patients with untreated goitrous Graves' disease but also from 14 patients with goitrous Hashimoto's thyroiditis elicited an increase in the N/C index, which exceeded the mean +2 S.D. of the values for 17 normal subjects (mean +/- S.D., 1.063 +/- 0.014). Four patients with primary myxedema displayed a normal N/C index. In Graves' disease, the N/C index did not correlate significantly with thyroid stimulating antibodies (TSAb) activities but did correlate significantly with estimated goiter size (P < 0.05). IgGs containing blocking-type TSH-receptor antibodies inhibited the TSH- or Graves IgG-stimulated increase in N/C index almost completely. but did not influence the stimulatory effect of IgG from two patients with Hashimoto's thyroiditis. CONCLUSIONS: We have developed a sensitive and simple assay for thyroid growth stimulating activity by using CB, and found that all tested patients with goitrous Graves' disease and goitrous Hashimoto's thyroiditis have thyroid growth stimulating immunoglobulins whose activity does not correlate with TSAb. Record Date Created: 19970710

18/7/45 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 09159599 97154483 PMID: 9001201

Thyroid hormones as neurotransmitters.

Dratman MB; Gordon JT

Department of Medicine, MCP Hanneman School of Medicine, Allegheny University, and Medical Research Service, Veterans Affairs Medical Center, Philadelphia, Pennsylvania 19104, USA. Thyroid (UNITED STATES) Dec 1996, 6 (6) p639-47, ISSN 1050-7256 Journal Code: BJW Contract/Grant No.: 45252, PHS Languages: ENGLISH Document type: Journal Article; Review; Review, Tutorial Record type: Completed

During brain development, before the apparatus of neurotransmission has been set into place, many neurotransmitters act as growth regulators. In adult brain, their role in neurotransmission comes to the fore but neuronal plasticity and other growth-related processes are their continuing responsibility. This has been clearly demonstrated for catecholamines. Previous as well as recent evidence now indicates that thyroid hormones may participate in the developing and adult brain through similar mechanisms. Immunohistochemical mapping of brain triiodothyronine (antibody specificity established by numerous appropriate tests) demonstrated that the hormone was concentrated in both noradrenergic centers and noradrenergic projection sites. In the centers (locus coeruleus and lateral tegmental system) triiodothyronine staining, like that of tyrosine hydroxylase, was heavily concentrated in cytosol and cell processes. By contrast, in noradrenergic targets, label was most prominent in cell nuclei. Combined biochemical and morphologic data allows a construct of thyroid hormone circuitry to unfold: The locus coeruleus is conveniently located just beneath the ependyma of the 4th ventricle. Thyroxine, entering the brain via the choroid plexus, is preferentially delivered to subependymal brain structures. High concentrations of locus coeruleus norepinephrine promote active conversion of thyroxine to triiodothyronine, leading to the preeminence of the locus coeruleus as a site of triiodothyronine concentration. Results of treatment with the locus coeruleus neurotoxin DSP-4 established that axonal transport accounts for delivery of both triiodothyronine and norepinephrine from locus coeruleus to noradrenergic terminal fields. The apparatus for transduction of thyronergic and noradrenergic signals at both membrane and nuclear sites resides in the postsynaptic target cells. Upon internalization of hormone in post-synaptic target cells, genomic effects of triiodothyronine, norepinephrine, and/or their second messengers are possible and expected. The evidence establishes a direct morphologic connection between central thyronergic and noradrenergic systems, supporting earlier proposals that triiodothyronine or its proximate metabolites may serve as cotransmitters with norepinephrine in the adrenergic nervous system. (35 Refs.) Record Date Created: 19970328

18/7/84 DIALOG(R)File 155:MEDL (c) format only 2001 Dialog Corporation. All rts. reserv. 08164191 94255188 PMID: 819692

Control of eyelid retraction associated with Graves' disease with botulinum A toxin. Biglan AW

Department of Ophthalmology, University of Pittsburgh School of Medicine, Pa. Ophthalmic surgery (UNITED STATES) Mar 1994, 25 (3) p186-8, ISSN 0022-023X Journal Code: OIC Languages: ENGLISH Document type: Journal Article Record type:

Two patients had satisfactory control of eyelid retraction associated with thyroid orbitopathy with repeated treatment of the levator palpebrae superioris muscle with botulinum A toxin. The effects of the toxin lasted for 3 to 4 months. Record Date Created: 19940630

18/7/88 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 08072229 93228655 PMID: 8471065

Tissue- and subunit-specific regulation of G-protein expression by hypo- and hyperthyroidism. Michel-Reher MB; Gross G; Jasper JR; Bernstein D; Olbricht T; Brodde OE; Michel MC Department of Medicine, University of Essen, Germany.

Biochemical pharmacology (ENGLAND) Apr 6 1993, 45 (7) p1417-23, ISSN 0006-2952 Journal Code: 9Z4 Contract/Grant No.: HL 38741, HL, NHLBI Languages: ENGLISH Document type: Journal Article Record type: Completed

Thyroid hormone status has profound effects on signal transduction in various tissues throughout the body. Therefore, we quantified the signal transducing G-proteins in the rat heart, cerebral cortex, vas deferens and liver by immunoblotting and pertussis toxin labeling in response to chemically induced hypothyroidism (treatment with propylthiouracil) and hyperthyroidism (treatment with triiodothyronine). Levels of the pertussis toxin (PTX) substrates Gi alpha and Go alpha in the heart and vas deferens were inversely correlated with thyroid hormone levels, i.e. Gi alpha and Go alpha were decreased or unchanged in hyperthyroid rats and increased in hypothyroid rats compared to control animals. The cerebral cortex and liver expression of PTX substrates Gi alpha and Go alpha was not affected by changes in thyroid hormone. Regulation of Gs alpha protein was more complex in that Gs alpha was unaffected in the other tissues tested. Expression of G-protein beta-subunits was not affected by thyroid status in the heart, liver, or cerebral cortex. Our results suggest that tissue- and G-protein-specific factors are involved in the regulation of G-protein subunits by thyroid hormone. Moreover, cardiac expression of Gs alpha is upregulated by increases or decreases in the normal level of thyroid hormone. Record Date Created: 19930510

18/7/93 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 07947261 94041171 PMID: 8225200

Regulation of calcitonin secretion in vitro.

Raue F; Zink A; Scherubl H

Abt. Innere Medizin I, Endokrinologie und Stoffwechsel, Universität Heidelberg, Germany. Hormone and metabolic research (GERMANY) Sep 1993, 25 (9) p473-6, ISSN 0018-5043 Journal Code: GBD Languages: ENGLISH Document type: Journal Article; Review, Review, Tutorial Record type: Completed

The concentration of extracellular calcium rightly regulates calcitonin secretion by calcium influx through dihydropyridine-sensitive voltage-dependent calcium channels; the result is an increase in intracellular calcium. There also exists a cAMP-dependent pathway of calcitonin release activated by glucagon or growth hormone releasing hormone. In thyroid C-cells, as in all cells, there is dual regulation of adenylate cyclase, mediated by inhibitory or stimulatory G proteins; glucagon stimulated cAMP production can be inhibited by somatostatin via pertussis toxin sensitive inhibitory G proteins. Somatostatin inhibits not only cAMP dependent but also calciumdependent calcitonin secretion. Furthermore, somatostatin inhibits voltage dependent calcium channel currents thereby lowering cytosolic calcium. These actions also involve a pertussis toxin sensitive inhibitory G protein but they occur independently of changes in the cytosolic cAMP concentration. Thus multiple interactions between second messenger systems at different cellular levels modulate calcitonin secretion. (30 Refs.) Record Date Created: 19931217

18/7/94 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 07911503 93145899 PMID: 8381075

Overexpression of the intact thyrotropin receptor in a human thyroid carcinoma cell line. Namba H; Yamashita S; Usa T; Kimura H; Yokoyama N; Izumi M; Nagataki S Department of Cell Physiology, Nagasaki University School of Medicine, Japan. Endocrinology (UNITED STATES) Feb 1993, 132 (2) p839-45, ISSN 0013-7227 Journal Code: EGZ Languages: ENGLISH Document type: Journal Article Record type: Completed Although thyrotropin is known to regulate thyroid cell differentiation and proliferation, human thyroid carcinoma cells are relatively insensitive or resistant to TSH stimulation. The expression levels of TSH receptor are significantly lower in carcinoma tissues than in normal tissues. Furthermore, in vitro human thyroid cell growth is not regulated by TSH itself. We, therefore, isolated neomycin-resistant stable human thyroid carcinoma cell (WRO cell) transfectants overexpressing intact human TSH receptor to evaluate the functional role of TSH receptor on carcinoma cells. Southern blot analysis confirmed incorporation and amplification of human TSH receptor complementary DNA sequences into genomic DNA. Northern gel analysis and reverse transcriptase-polymerase chain reaction analysis revealed the presence of specific TSH receptor messenger RNA (4.0 kilobases), and the specific binding and the affinity of [125I]TSH on stably transfected WRO cells were demonstrated compared to wild type. Nevertheless, impaired cAMP production to transfectants by TSH was observed, cAMP production was confirmed after stimulation of both wild type and transfectants by forskolin, cholera toxin, and isoproterenol. In contrast, TSH could affect the cytoplasmic calcium mobilization immediately after the addition of TSH to WRO transfectants. These results suggest that the impairment of TSH action on human thyroid carcinoma cells is not due to a major structural abnormality of the TSH receptor, reduction

in the receptor number, or receptor affinity, but much more likely du nucleotide-binding protein coupling defect. Record Date Created: 302 20/6/1 10358931 20000559 PMID: 10532769 Strabismus surgery among aged medicare beneficiaries. Dec 1997

20/6/2 10021711 99082623 PMID: 9865104
[Bilateral taryngeal movement disorder and synkinesia: value of botulism toxin. Apropos of a case] Trouble de la mobilite laryngee bilaterale et syncinesies: interet de la toxine botulique. A propos d'un cas. 1998

20/6/3 08164191 94255188 PMID: 8196925
Control of eyelid retraction associated with Graves' disease with botulinum A toxin, Mar 1994

20/6/4 07433158 91214931 PMID: 1902375 Management of dysthyroid eye disease. Apr 1991

20/6/5 07111148 94157131 PMID: 8113438 Botulinum toxin type A in upper lid retraction of Graves' ophthalmopathy. Dec 1993

20/6/6 06668510 91032399 PMID: 2226978 Thyroid eye disease. 1990

20/6/7 06668494 91032402 PMID: 2226981 Botulinum toxin therapy in dysthyroid strabismus. 1990

20/6/8 06275988 87108295 PMID: 3804629 Botulinum in the treatment of adult motility disorders. Winter 1986

20/6/9 06220002 86204801 PMID: 3703521

Botulinum toxin for the treatment of dysthyroid ocular myopathy. Apr 1986

20/6/10 06145851 86084827 PMID: 3841096 Botulinu m chemodenervation for strabismus and other disorders. Winter 1985

20/6/11 05312485 89385497 PMID: 2779991 Botulinum toxin therapy of eye muscle disorders. Safety and effectiveness. American Academy of Ophthalmology. Sep 1989

20/6/12 05259551 90199325 PMID: 3273259 [The use of botulinum toxin in endocrine exophthalmos] Utilisation de la toxine botulinique dans les exophtalmies endocriniennes. 1988

20/6/13 05204825 89026356 PMID: 3179055 Botulinum toxin. Aug 1988

20/6/14 05131078 87070940 PMID: 3466462 Diplopia in thyroid eye disease. 1986

20/6/15 04905297 84225515 PMID: 6676980 Saccadic velocity measurements in strabismus. 1983

20/6/16 04576471 85026678 PMID: 6489104 Injection treatment of endocrine orbital myopathy. Aug 15 1984

20/7/6 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 06668510 91032399 PMID: 2226978

Thyroid eye disease.

Elston JS

Eye (ENGLAND) 1990, 4 (Pt 4) pvii, ISSN 0950-222X

Journal Code: EYE

Kowal L 24/7/1 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 07433158 91214931 PMID: 1902375

Management of dysthyroid eye disease.

Fells P

Moorfields Eye Hospital, London.

British journal of ophthalmology (ENGLAND) Apr 1991, 75 (4) p245-6, ISSN 0007-1161 Journal Code: AZK Languages: ENGLISH Document type: Journal Article: Review: Review. Tutorial Record type: Completed (10 Refs.) Record Date Created: 19910605

24/7/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation, All rts, reserv. 06668510 91032399 PMID: 2226978

Thyroid eye disease.

Elston JS

Eye (ENGLAND) 1990, 4 (Pt 4) pvii, ISSN 0950-222X Journal Code: EYE Languages: ENGLISH Document type: Editorial Record type: Completed Record Date Created: 19901205

24/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 06668494 91032402 PMID: 2226981

Botulinum toxin therapy in dysthyroid strabismus.

Lyons CJ; Vickers SF; Lee JP

Moorfields Eye Hospital, London.

Eye (ENGLAND) 1990, 4 (Pt 4) p538-42, ISSN 0950-222X Journal Code: EYE Languages: ENGLISH Document type: Journal Article Record type: Completed We report our experience with the use of Botulinum toxin injection in 38 patients (64 injections) with severe dysthyroid strabismus. Three quarters of the injections led to a decrease in the angle of the squint by a mean 75% of the initial deviation. The average duration of effect was two months. Twenty six patients went on to surgery after stabilisation of their squint and endocrine status. Six patients achieved a stable long-term result with Botulinum toxin only. We suggest these results of treatment of early dysthyroid myopathy are more consistent with the characteristics of inflammatory spasm than contracture. The value of Botulinum toxin as a temporary means of maintaining binocularity in these young patients is discussed. Record Date Created: 19901205

24/7/4 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 06275988 87108295 PMID: 3804629

Botulinum in the treatment of adult motility disorders.

Hoffman RO; Helveston EM

International ophthalmology clinics (UNITED STATES) Winter 1986, 26 (4) p241-50, ISSN 0020-8167 Journal Code: GTZ Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19870326

24/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 06220002 86204801 PMID: 3703521

Botulinum toxin for the treatment of dysthyroid ocular myopathy.

Dunn WJ; Arnold AC; O'Connor PS

Ophthalmology (UNITED STATES) Apr 1986, 93 (4) p470-5, ISSN 0161-6420 Journal Code: OI5 Languages: ENGLISH Document type: Journal Article Record type: Completed Eight consecutive patients with acquired deviations due to dysthyroid ocular myopathy were injected with botulinum A toxin for relief of their diplopia. Seven patients were acute in the onset of symptoms and one was chronic. All showed improvement in motility and experienced a reduction if not total relief of their symptoms. Six patients required reinjection. Complications were limited to transient ptosis, transient involvement of adjacent muscles and transient but prolonged paralysis that eventually resolved. No systemic complications were noted. We conclude that chemodenervation with botulinum A toxin may have a role in the management of dysthyroid ocular myopathy not amenable to prism treatment and may act as an adjunct to or eliminate the need for surgical correction in some patients. Record Date Created: 19860613

24/7/6 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 05204825 89026356 PMID: 3179055

Botulinum toxin.

Kowal L

Australian and New Zealand journal of ophthalmology (AUSTRALIA) Aug 1988, 16 (3) p264-6, ISSN 0814-9763 Journal Code: ANZ Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19881220

24/7/7 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 05131078 87070940 PMID: 3466462

Diplopia in thyroid eye disease.

Fells P; McCarry B

Transactions of the ophthalmological societies of the United Kingdom (ENGLAND) 1986, 105 (Pt 4) p413-23, ISSN 0078-5334 Journal Code: WA1 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19870122

ages: ENGLISH Document type: Editorial Record Completed Record Date Created: 19901205

20/7/13 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation, All rts, reserv. 05204825 89026356 PMID: 3179055 Botulinum toxin.

an and New Zealand journal of ophthalmology ALIA) Aug 1988, 16 (3) p264-6, ISSN 0814-9763 Journal Code: ANZ Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19881220

24/7/8 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv. 04576471 85026678 PMID: 6489104

Injection treatment of endocrine orbital myopathy.

Scott AB

Documenta ophthalmologica (NETHERLANDS) Aug 15 1984, 58 (1) p141-5, ISSN 0012-4486 Journal Code: EBF

Contract/Grant No.: EY02106, EY, NEI Languages: ENGLISH Document type: Journal Article Record type: Completed

Eight patients with endocrine orbital myopathy received botulinum toxin injection of extraocular muscles for strabismus or injections of the levator for lid retraction. Strabismus of 25 prism iopters or less, especially during early stages of eye muscle involvement, responded well to injection with realignment and, probably, with avoidance of fixed muscle shortening. Long-standing strabismus, large angles, and lid retraction responded less well. Record Date Created: 19841123